

Curcumin attenuates oxidative damage in animals treated with a renal carcinogen, ferric nitrilotriacetate (Fe-NTA): Implications for cancer prevention

Abstract

Curcumin (diferuloylmethane), a biologically active ingredient derived from rhizome of the plant *Curcuma longa*, has potent anticancer properties as demonstrated in a plethora of human cancer cell lines/ animal carcinogenesis model and also acts as a biological response modifier in various disorders. We have reported previously that dietary supplementation of curcumin suppresses renal ornithine decarboxylase (Okazaki et al. *Biochim Biophys Acta* 1740:357-366, 2005) and enhances activities of antioxidant and phase II metabolizing enzymes in mice (Iqbal et al. *Pharmacol Toxicol* 92:33-38, 2003) and also inhibits Fe-NTA-induced oxidative injury of lipids and DNA in vitro (Iqbal et al. *Teratog Carcinog Mutagen* 1:151-160, 2003). This study was designed to examine whether curcumin possess the potential to suppress the oxidative damage caused by kidney-specific carcinogen, Fe-NTA, in animals. In accord with previous report, at 1 h after Fe-NTA treatment (9.0 mg Fe/kg body weight intraperitoneally), a substantial increased formation of 4-hydroxy-2-nonenal (HNE)-modified protein adducts in renal proximal tubules of animals was observed. Likewise, the levels of 8-hydroxy-2-deoxyguanosine (8-OHdG) and protein reactive carbonyl, an indicator of protein oxidation, were also increased at 1 h after Fe-NTA treatment in the kidneys of animals. The prophylactic feeding of animals with 1.0% curcumin in diet for 4 weeks completely abolished the formation of (i) HNE-modified protein adducts, (ii) 8-OHdG, and (iii) protein reactive carbonyl in the kidneys of Fe-NTA-treated animals. Taken together, our results suggest that curcumin may afford substantial protection against oxidative damage caused by Fe-NTA, and these protective effects may be mediated via its antioxidant properties. These properties of curcumin strongly suggest that it could be used as a cancer chemopreventive agent. © Springer Science+Business Media, LLC. 2009.